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THE ASSESSMENT OF EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

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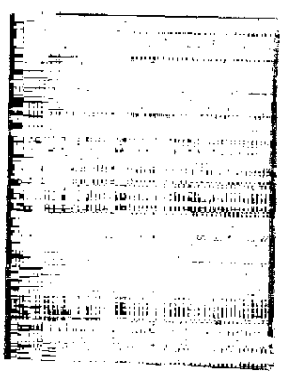
This paper examines the manner in which assessments of exposure to environmental tobacco smoke (ETS) are used in epidemiological studies seeking to establish an association between such exposure and diseases such as lung cancer. Two such studies are described. A review of the literature shows that exposure to ETS is not accurately measured by subjective evaluations communicated via questionnaire. The inaccuracy is evident in situations where recent exposure is being measured and may reasonably be expected to be even greater when long-term exposure is at issue. Such inaccuracies do not necessarily invalidate the conclusions that may be drawn from a case/control study. In a situation where all types of errors occur equally frequently among cases and controls, the effects of such errors are to lower the relative risk estimate, to reduce the power of the study, and to make it more difficult to establish an association between exposure and disease. However, in a situation where the pattern of errors is different for cases than it is for controls, there is considerable potential for the errors to produce misleading conclusions. Increased concentration on the validation of subjective ETS exposure assessments is recommended. *Copyright © 1997 Elsevier Science Ltd*



INTRODUCTION

In recent years, much attention has been focussed on the potential health effects of exposure to environmental tobacco smoke (ETS). In 1986, reports by the U.S. Public Health Service (1986) and by the National Research Council (1986) of the National Academy of Sciences reached similar conclusions about the adverse health effects of involuntary smoking on healthy adults and children. They concluded, for the first time, that the involuntary inhalation of cigarette smoke by nonsmokers can cause disease, most notably lung cancer. The 1992 report on the respiratory health effects of exposure to ETS, issued by the U.S. Environmental Protection Agency (USEPA 1992), concluded that ETS is a human lung carcinogen, responsible for approximately 3000 lung cancer deaths per year in U.S. nonsmokers. These conclusions were based primarily on epidemiological evidence from a small number of prospective studies and a larger number of case/control retrospective studies. The methodology used in all these studies involved a comparison between a group of nonsmoking subjects with the disease of interest and a group of nonsmoking subjects who did not have the disease of interest in terms of exposure to ETS over a particular period of time. In the context of diseases such as lung cancer, the relevant period of time was the subject's lifetime up to the time of diagnosis.

There are many methodological problems associated with epidemiological studies of this kind. Thornton et al. (1994) demonstrated that, for many lifestyle risk factors, prevalence is higher among people who live with a smoker. Analyses which do not control for such

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confounding risk factors are potentially misleading. The possibilities of bias due to misclassification of smokers as nonsmokers were documented by Lee (1988). In this paper, the difficulties involved in obtaining accurate data concerning a subject's exposure to ETS are examined. In studies where the health impact of long-term exposure to ETS is at issue, the data on exposure are usually collected by way of questionnaire. In studies of the effects of short-term exposure, exposure data collected by way of questionnaire are often supplemented by measurements of biomarkers for nicotine such as cotinine.

VALIDATION OF EXPOSURE ASSESSMENTS

The first step in any investigation of the health effects of exposure to ETS is to define a scale on which the extent of exposure is to be recorded. Three types of scale are commonly used: 1) a dichotomous scale representing the presence or absence of exposure from a specific source; 2) an ordinal scale corresponding to increasing levels of exposure; and, 3) a quantitative scale corresponding to the exact level of exposure. An example of a dichotomous scale would be the presence or absence of exposure from a spouse. An ordinal scale might be an assessment of exposure from a spouse on a scale of None, Low, Medium, and High. A quantitative scale might be the number of years spent living with a spouse who smoked.

The second step in an investigation is to define a methodology whereby the placement of each subject on the particular scale defined may be estimated. Such placements may be accomplished using questionnaire assessments by the subject or by a surrogate. When exposure refers to a recent time period of relatively short duration, exposure assessments may be obtained from measurements of biomarkers for nicotine such as cotinine. There is currently no biomarker available for lifetime exposure.

The estimated placement E^* for a subject may differ from the subject's true placement E. The primary interest is in the relationship between disease status and E. The data allow investigation of the relationship between disease status and E^*. The extent to which conclusions reached regarding the relationship between disease status and E^* may also apply to the relationship between disease status and E must be examined.

Dichotomous and ordinal scales

In this section, dichotomous and ordinal scales are considered. Suppose that the scale in question has M levels referred to as 1, 2,..., M in such a way that level 1 implies a lower level of exposure than level 2 and so on. A dichotomous scale corresponds to the case M = 2. A misclassification occurs, for example, if a subject whose true placement is $E = 2$ is given an estimated placement of $E^* = 1$.

In situations where misclassifications occur at random and independently of disease status, the association between disease status and E^* tends to attenuate the association between disease status and E (Gladen and Rogan 1979; Kraemer 1985). Suppose that the following table represents the true cross-classification of subjects by case/control status and exposure status:

	Cases	Controls	Total
$E = 2$	400	380	780
$E = 1$	200	220	420
Total	600	600	1200

The table yields an odds ratio of 1.16. Now suppose that, for both cases and controls, 5% of subjects with $E = 1$ are misclassified so that $E^* = 2$. Then the observed table will be:

	Cases	Controls	Total
$E^* = 2$	420	402	822
$E^* = 1$	180	198	378
Total	600	600	1200

This table yields an odds ratio of 1.15, which is closer to the null value of 1 than was the odds ratio calculated from the true exposure data.

Kilpatrick (1987) raised the possibility of differential recall bias in situations where the rate and direction of misclassification depends on the subject's disease status. An association between disease status and E^* may come about as a result of over-reporting of exposure among cases and/or under-reporting of exposure among controls. As an example, suppose that the true cross-classification of subjects by case/control status and exposure status is as before and that misclassifications occur in such a way that 10% of cases with $E = 1$ are recorded as having $E^* = 2$, while 10% of controls with $E = 2$ are recorded as having $E^* = 1$. Then the observed table will be:

	Cases	Controls	Total
$E^* = 2$	420	342	762
$E^* = 1$	180	258	438
Total	600	600	1200

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This table yields an odds ratio of 1.76, which is much larger than the odds ratio of 1.16 calculated from the true exposure data.

Barry (1996) also considered differential recall bias and proposed methods whereby the sensitivity of conclusions regarding the association between disease and E^* to specified levels of recall bias may be established. In evaluating the methodology which is used to produce E^* as an estimate of E, a quantity of interest would be, for example, the proportion of subjects having $E = 2$ that are estimated to have $E^* = 1$. Such proportions will be referred to as misclassification rates. Information regarding the misclassification rates for both cases and controls is required. Given these misclassification rates, the methods of Barry (1996) can be used to correctly infer the relationship between disease status and E from that observed between disease status and E^*. Without these misclassification rates, the possibility can never be ruled out that a relationship found to exist between disease status and reported exposure E^* is no more than an artifact produced by differential recall bias.

Quantitative scales and logistic regression

In this section, the use of quantitative scales in logistic regression analysis is considered. Define $P(x)$ to be the probability of disease for a subject whose true exposure is $E = x$. Then the log odds on being diseased is:

$$LO(x) = \log[\frac{P(x)}{1-P(x)}]$$

The logistic regression model assumes that:

$$LO(x) = \alpha + \beta x$$

for some values of α and β. A value of $\beta = 0$ implies that $LO(x)$ (and hence $P(x)$) does not depend on the value of x. A value of $\beta > 0$ implies that $LO(x)$ (and hence $P(x)$) is an increasing function of x or, in other words, that disease is more likely for larger exposure values. Data for which the null hypothesis that $\beta = 0$ is rejected are used to establish an association between exposure and disease. Barry (1996) showed that the score test of the null hypothesis that $\beta = 0$ is equivalent to Student's two-sample t-test comparing the mean exposure level of cases with that of controls.

In most cases, it is not possible to obtain true exposure levels and analyses must proceed using reported or measured exposure levels. Suppose that the measured exposure E^* is related to the true exposure E according to the relationship:

$$E^* = E + \epsilon$$

where ϵ represents a normally distributed error with mean μ and standard deviation σ. Rosner et al. (1989) showed that analyses carried out by assuming that the observed exposure measurement E^* is identical to the true exposure E are biased towards acceptance of the null hypothesis that $\beta = 0$, i.e., the null hypothesis of no association. These results assume that the same error mechanism operates for both cases and controls. The extent of the bias is crucially dependent on the correlation between true exposure and measured exposure. In fact, if T denotes the value of Student's two-sample t-statistic for comparing the mean true exposure of cases with that of controls and T^* denotes the same quantity for measured exposure, then $T^* \approx \rho T$ where ρ is the correlation coefficient between true exposure and measured exposure.

Differential recall bias may also be a factor in logistic regression analyses. Barry (1996) has shown that the probability of finding an association between disease and estimated exposure E^* is increased by recall error mechanisms having one or more of the following effects: 1) over-estimation of the mean exposure level for cases; 2) under-estimation of the mean exposure for controls; and, 3) under-estimation of the variability of exposure among cases and/or controls. Validations of exposure assessment methods should seek to eliminate these effects as explanations for any associations discovered.

Quantitative scales and the creation of ordinal scales

The range of a quantitative scale is often split into intervals to define an ordinal scale. If the measured exposure is not identical to the true exposure then errors on the quantitative scale will translate into errors on the derived ordinal scale.

The intervals for the ordinal scale are often selected so that the percentages of subjects falling in each interval are equal. In situations where the exposure measurement errors follow a normal distribution, the percentage agreement (PA) between the ordinal scale derived from E^* and that derived from E may be determined from the value of the correlation coefficient ρ between E^* and E. Consider an ordinal scale with two levels corresponding to below average exposure and above average exposure, respectively. The following

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table demonstrates the dependence on ρ of the PA between the ordinal scale derived from E^* and that derived from E:

ρ	0.0	0.1	0.2	0.3	0.4
PA	50	53	56	60	63
ρ	0.5	0.6	0.7	0.8	0.9
PA	67	71	75	80	86

Thus, for example, if the correlation between E^* and E is 0.7, then 75% of subjects will have the same placement on the ordinal scale derived from E^* as they would have on the ordinal scale derived from E. Table I considers an ordinal scale with four levels corresponding to a division of the exposure range into quartiles. The table shows, for various values of ρ, the distribution among the quartiles of measured exposure E^* of subjects in each quartile of true exposure E. Consider ρ = 0.7. Then 60% of subjects in the first quartile of true exposure will fall in the first quartile of measured exposure, 27% in the second, 11% in the third, and 2% in the fourth. The corresponding values are 27%, 35%, 27%, and 11% for subjects in the second quartile of true exposure; 11%, 27%, 35%, and 27% for subjects in the third quartile; and 2%, 11%, 27%, and 60% for subjects in the fourth quartile. Clearly the misclassification rates are substantial even for a quite large value for ρ.

The use of ordinal scales derived from quantitative scales may lead to differential recall bias if the error mechanism involved in the creation of E^* from E is different for cases than it is for controls. For instance, if the average value of $E^* - E$ tends to be higher for cases than for controls, then cases will tend to be placed too high up and controls too low down on the derived ordinal scale. A spurious association between the ordinal scale and case/control status results.

Questions to ask

When evaluating a particular methodology used to estimate the placement of subjects on an exposure scale, the following questions should be addressed:

Ordinal scales:

1) Are estimates of the misclassification rates presented?

2) Do the same misclassification rates apply to both cases and controls? If not, has the analysis been corrected for differential recall bias?

Quantitative scales:

1) Have estimates of the correlation between true exposure and measured exposure been presented?

2) Is there a tendency for measured exposure to either over-estimate or under-estimate true exposure?

3) Does the variability in measured exposure values reflect the variability in true exposure values?

4) Does the same error mechanism apply to both cases and controls? If not, has the analysis been corrected for differential recall bias?

THE FONTHAM STUDY

Fontham et al. (1994) described a case/control study designed to determine the relative risk of lung cancer in lifetime never smokers associated with ETS exposure. Eligible cases consisted of female nonsmoking residents of five metropolitan areas of the U.S. with microscopically confirmed primary carcinoma of the lung who were diagnosed between 1 December 1986 and 30 November 1988. A population based control group was selected by random digit dialling and supplemented by random sampling from the Health Care Financing Administration files for women 65 years and older. Controls were frequency matched to cases on race and age in a 2:1 ratio of controls to cases and met the same residence and personal tobacco use criteria as cases. A lifetime history of exposure to ETS was obtained via questionnaire. The questionnaire was completed by all 1253 controls and by 412 of the 653 cases. The questionnaires for the remaining 241 cases were completed by the next of kin.

The paper reported results for various exposure measures derived from the questionnaire data. Exposure to ETS was examined by source during childhood (father, mother, and other household members who lived in the home for at least 6 months) and during adult life (spouse, other household members, occupational and social exposures). Childhood included the years from birth through age 18 y. Exposures from parents after that time were classified as other household members during adult life. Dichotomous ETS exposure (ever and never) was examined by source and type of tobacco. Pack-years of cigarette smoke exposure from spouse were calculated by multiplying the number of packs smoked per day by the number of years the spouse smoked cigarettes while living with the study subject.

Analyses are presented for dichotomous (ever and never) exposure measures, polychotomous exposure measures based on sub-divisions of the range of

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Table 1. For a quantitative exposure scale, the table shows, for various values of the correlation ρ between true exposure E and measured exposure E^*, the distribution among the quartiles of E^* of subjects in each quartile of E.

ρ = 0.0		Estimated			
		1	2	3	4
True	1	25	25	25	25
	2	25	25	25	25
	3	25	25	25	25
	4	25	25	25	25

ρ = 0.5		Estimated			
		1	2	3	4
True	1	48	28	17	7
	2	28	29	26	17
	3	17	26	29	28
	4	7	17	28	48

ρ = 0.1		Estimated			
		1	2	3	4
True	1	29	26	24	21
	2	26	25	25	24
	3	24	25	25	26
	4	21	24	26	29

ρ = 0.6		Estimated			
		1	2	3	4
True	1	54	28	14	4
	2	28	32	26	14
	3	14	26	32	28
	4	4	14	28	54

ρ = 0.2		Estimated			
		1	2	3	4
True	1	33	27	23	17
	2	27	26	25	22
	3	22	25	26	27
	4	17	23	27	33

ρ = 0.7		Estimated			
		1	2	3	4
True	1	60	27	11	2
	2	27	35	27	11
	3	11	27	35	27
	4	2	11	27	60

ρ = 0.3		Estimated			
		1	2	3	4
True	1	38	27	21	14
	2	27	27	25	21
	3	21	25	27	27
	4	14	21	27	38

ρ = 0.8		Estimated			
		1	2	3	4
True	1	68	25	6	1
	2	25	41	27	7
	3	7	27	41	25
	4	1	6	25	68

ρ = 0.4		Estimated			
		1	2	3	4
True	1	43	28	19	10
	2	28	28	25	19
	3	19	25	28	28
	4	10	19	28	43

ρ = 0.9		Estimated			
		1	2	3	4
True	1	77	21	2	0
	2	21	52	25	2
	3	2	25	52	21
	4	0	2	21	77

variables such as pack-years of exposure from spouse and childhood smoke-years of exposure, and logistic regressions (Breslow and Day 1980) using the actual values of variables such as pack-years of exposure from spouse. The analyses are adjusted for the potential confounding variables age, race, study area, education, intake of fruits and vegetables, supplemental vitamin index, dietary cholesterol, family history of lung cancer, and employment in high risk occupations.

Consider first a measure of exposure which defines a subject as having been exposed if the subject ever

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lived with a spouse who smoked and unexposed otherwise. The following is the relevant 2 x 2 table:

	Cases	Controls	Total
Exposed	433	766	1199
Unexposed	218	487	705
Total	651	1253	1904

The table yields a crude odds ratio of $OR = 1.26$ with 95% confidence interval [1.04, 1.54]. The odds ratio adjusted for the potential confounding variables age, race, study area, education, intake of fruits and vegetables, supplemental vitamin index, dietary cholesterol, family history of lung cancer, and employment in high risk occupations, is $OR = 1.29$ with 95% confidence interval [1.04, 1.60].

Now, the quantitative measure of exposure to ETS defined as pack-years of cigarette smoke exposure from the spouse is considered. Following are the results comparing all 609 lung cancer carcinoma cases for whom data were available with the 1205 controls for whom data were available when subjects are divided into five groups based on reported exposure:

Pack-y	Cases	Controls	*OR* (P-value)	OR^a (P-value)
0	267	562		
0 - 15	146	300	1.02 (0.438)	1.08 (0.279)
15 - 40	92	190	1.02 (0.447)	1.04 (0.403)
40 - 80	80	126	1.34 (0.033)	1.36 (0.038)
≥ 80	24	27	1.87 (0.016)	1.79 (0.028)

The odds ratios quoted are those comparing the particular exposure group with the group reporting no exposure. The odds ratios written as OR^a are adjusted for the potential confounders listed earlier. The p-values are those for a one-sided test of the null hypothesis that the true odds ratio is equal to 1. A test for trend based on an unconditional logistic regression analysis yielded a p-value of 0.015 whether or not the potential confounders were included in the model.

Subjects may be divided into four groups based on presence/absence of adult exposure and presence/ absence of childhood exposure. The following table cross classifies subjects by group and case/control status:

Exposed as child	Exposed as adult	Cases	Controls	*OR*	P-value
No	No	33	71	1.00	
No	Yes	182	365	1.07	0.380
Yes	No	8	44	0.39	0.986
Yes	Yes	305	725	0.91	0.674

The odds ratios quoted above compare the particular group with the group who experienced no exposure either as children or as adults.

This table did not feature in either of the published reports of the Fontham study. Instead, the authors reported the effects of adult exposure separately for subjects who experienced childhood exposure and for those who did not. For subjects who did not experience childhood exposure, the odds ratio for adult exposure was 1.07 with a one-sided p-value of 0.380. For subjects who did experience childhood exposure, the odds ratio for adult exposure was 2.31 with a one-sided p-value of 0.014. The authors concluded that the "risk is more marked for women who have also been exposed to ETS during childhood". This conclusion fails to take into account the fact that subjects with childhood but no adult ETS exposure are at a lower risk of lung cancer compared to subjects with neither childhood nor adult exposure. This anomaly may be an artifact of bias either in study design or data collection.

The authors conclude that long-term exposure to ETS increases risk of lung cancer in women who have never personally used tobacco and the risk is increased for those who were exposed to ETS during childhood.

Justification of these conclusions requires validation of the exposure assessment instruments used in the study. In the next section, the extent of such validation in the Fontham study is assessed.

VALIDATION OF MEASUREMENTS OF EXPOSURE TO ETS

Validation in the Fontham Study

There is no indication in either of the reports emanating from the Fontham study (Fontham et al. 1991; 1994) that any formal validation of the methodology used to assess exposure to ETS was carried out. However, it is clear that the authors took a number of steps to ascertain the extent of differential recall bias.

In the first three years of the study, two control groups, one with colon cancer and one from the general population, were selected for case/control comparisons.

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It was hoped that differential recall bias between cases and colon cancer controls would be minimized since both groups are similarly motivated to recall earlier exposure. (This hope may not be entirely justified since there has been widely publicized interest in the ETS/lung cancer relationship but not in the ETS/colon cancer relationship.) It was found in Fontham et al. (1991) that the results were consistent for case/control comparisons using each control group. The use of a colon cancer control group was not extended into the final two years of the study.

Separate analyses were conducted for subjects who personally responded and for subjects for whom information was obtained from surrogate respondents. Both analyses produced similar results.

The Fontham papers cited three references to questionnaire validation studies in the literature. Pron et al. (1988) reported a study in which a total of 117 control subjects, initially interviewed in a lung cancer case/control study conducted in Toronto, Canada, between 1983 and 1984, were reinterviewed on average six months later. Of the 117 respondents, 103 or 88% gave identical responses when asked if they were ever exposed to residential passive smoke. When questioned as to the smoking status of their spouses, 108 or 95% of 114 respondents gave identical responses. When questioned as to the smoking status of their mothers, 112 or 96% of 117 respondents gave identical responses. When questioned as to the smoking status of their fathers, 84 or 72% of 117 respondents gave identical responses. Of the 117 respondents, 57 or 49% gave identical responses as to the number of smokers (on a scale of 0, 1, 2, 3+) among people with whom they had shared living quarters. The correlation coefficient between pairs of reports of duration of exposure to passive smoke in the home was 0.45 based on 115 pairs. The correlation coefficient between pairs of reports of duration of exposure to passive smoke from the spouse was 0.25 based on 58 pairs.

Coultas et al. (1989) assessed the reliability of questionnaire responses on lifetime exposure to tobacco smoke in the home for a sample of 149 adult nonsmokers recruited in New Mexico in 1986. A structured questionnaire on lifetime exposure to ETS was administered by a trained interviewer to each subject on two occasions separated by approximately four to six months. All of the 67 subjects who, at the first interview, reported that their spouse smoked reported the same at the second interview. There was less agreement concerning the amount currently smoked by the spouse. When consumption was classified as less than one pack per day, one pack per day, and more than one pack per day, there was 43.8% agreement between the two interviews. There was a correlation of 0.95 between responses to the question: For how many years did your spouse smoke cigarettes while sharing your home? There was a correlation of 0.25 between responses to the question: On average, how many hours per day were you exposed to cigarette smoke from your spouse?

Riboli et al. (1990) considered a large international study in which 1369 nonsmoking women were interviewed. Thirteen centres located in ten countries took part in the study. The subjects were either control subjects from previous or ongoing case/control studies or were volunteers from stratified population samples. The results of the analysis of self-reported recent exposure to ETS from any source in relation to urinary concentrations of cotinine were presented. The authors calculated mean cotinine/creatinine levels for various subgroups of subjects. Of particular relevance is the comparison of the mean cotinine/creatinine levels across the four groups determined by the reported presence or absence of exposure in the home and in the workplace. The mean cotinine level was 2.7 for those who reported no exposure at either location, 4.8 for those who reported exposure at work but not at home, 9.0 for those who reported exposure at home but not at work, and 10.0 for those who reported exposure at both locations. No attempt was made to quantify the degree of overlap among the four groups in terms of cotinine levels. The authors also reported comparisons between the group of subjects who reported no exposure from any source and the group who reported exposure from all sources, i.e., home, work, and public places. The mean cotinine level was 3.0 for those who reported no exposure and 18.5 for those who reported exposure from all sources. Once again no attempt was made to quantify the degree of overlap between the two groups. The authors reported that the correlation coefficients between cotinine and cumulative duration of exposure were between 0.40 and 0.51 for nine centres, between 0.30 and 0.40 for two centres, and less than 0.30 for two centres.

No serious attempt was made to assess the validity of the questionnaire-based assessments of ETS exposure used in the Fontham study. The Riboli paper concerned recent exposure rather than lifetime exposure. All three of the studies cited reported validation studies carried out on healthy nonsmoking subjects and hence offered no indication of the possible extent of differential recall bias. Many of the

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misclassification rates quoted in the studies are substantial, especially in the context of quantitative exposure assessment. The correlation coefficients quoted may be unsatisfactory.

A more in-depth review of the literature dealing with validation of exposure assessment methodologies follows.

Validation of measurements of lifetime exposure to ETS

A literature search revealed a number of other studies (in addition to those cited by Fontham et al.) in which the validation of measurements of lifetime exposure to ETS was considered.

Rogot and Reid (1975) used data obtained from 1953 subjects included in the British-Norwegian Migrant Study to compare information on the smoking habits of an individual as reported by the individual himself some time before death with the responses on the same points given after the individual's death in a questionnaire addressed to his next-of-kin. Of the 1289 individuals who themselves claimed to be nonsmokers or occasional smokers, 76 or 6% were described as regular smokers by their next-of-kin; of the 664 individuals who themselves claimed to be regular smokers, 77 or 12% were described as nonsmokers or occasional smokers by their next-of-kin. These data represent a 92.2% agreement between subject and next-of-kin. Questions were also asked regarding the number of cigarettes smoked per day with answers being collapsed to none, <1 pack, and ≥1 pack. The following table shows the percentage distribution of next-of-kin responses for each possible subject response.

		Next-of-kin			
		None	<1	≥1	No. of subjects
Subject	None	91	6	3	1247
	<1	12	46	42	321
	≥1	3	14	83	385

The next-of-kin agreed with the subject's own response for 81.7% of subjects. Tobacco consumption was overstated by the next-of-kin for 12.8% of subjects and understated for 5.5%.

Kolonel et al. (1977) examined interview data on personal smoking habits when collected from two sources. A sample of 300 pairs of subjects (mostly husbands and wives) was obtained from an ongoing health survey in Hawaii, and both members of each pair were interviewed separately about the habits of the husband. Care was taken that the members of each pair had no opportunity to communicate with each other between the start and completion of both interviews. For the 300 pairs, there was agreement regarding whether the husband had ever smoked for 289 or 96.3% of the pairs. There were 194 husbands who smoked and for 166 of these data was available from both husband and surrogate concerning the number of cigarettes smoked per day. The mean for husbands was 20.9 and the mean for surrogates was 23.8. Of the pairs interviewed, 36.1% agreed exactly regarding the number of cigarettes smoked per day by the husband, 54.2% agreed to within plus or minus 5 cigarettes, and 78.3% agreed to within plus or minus 10 cigarettes.

Herrmann (1985) compared smoking data obtained from cases or controls and their respective next-of-kin as part of a study of colon cancer in the five Pennsylvania counties of the Philadelphia metropolitan area. The case population consisted of whites aged 45-69 y who had resided in the region for at least 2 y prior to diagnosis and who were diagnosed with colon cancer after 1 July 1976. Controls were selected using an area probability sampling scheme and were frequency matched to the case group. Herrmann reported that the percentage of complete agreement on whether the subject smoked exceeded 85% for both cases and controls. The agreement found between subject and next-of-kin when interviewer-administered questionnaires were used was slightly higher than that obtained when self-administered questionnaires were used. Both the subject and the next-of-kin were asked to state the numbers of cigarettes per day smoked by the subject. For 50 case/next-of-kin pairs, the correlation between the two reports was 0.83 with a mean absolute difference of 6.2. For 61 control/next-of-kin pairs, the correlation between the two reports was 0.77 with a mean absolute difference of 7.6.

In an attempt to assess the validity of the wife as a source of exposure information, Lerchen and Samet (1986) interviewed 80 wives in 1983-1984 for the same histories provided earlier by their husbands who were cases in a case/control study of lung cancer in New Mexico, 1980-1982. Wives correctly reported the cigarette smoking status of their husbands. The correlation coefficient between the husband's and wife's reports of the total years of smoking by the husband was 0.91 with the mean for husbands 46.1 and the mean for wives 46.8. The correlation coefficient between the husband's and wife's reports of the number of cigarettes smoked per day by the husband was 0.44 with the mean for husbands 27.6 and the mean for wives 27.9.

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Sandler and Shore (1988) reported a study of long-term effects of transplacental and childhood exposure to cigarette smoke, in which 518 cancer cases and 518 healthy controls were interviewed concerning parents' smoking habits during childhood and prior to birth. Parents or siblings of the study subjects were also interviewed to obtain the same information. There was 95.7% agreement between subjects and mothers as to whether the mother ever smoked cigarettes. There was 86.4% agreement between subjects and mothers as to whether the father ever smoked cigarettes. When the mother reported that she was not a smoker, 97% of the subjects agreed. When the mother reported that she was a smoker, 92% of the subjects agreed. When the mother reported that the father was not a smoker, 83% of the subjects agreed. When the mother reported that the father was a smoker, 88% of the subjects agreed. The authors reported no major differences between cases and controls in overall agreement between mothers and subjects on mothers' or fathers' smoking habits. They did, however, report some small differences between cases and controls for conditional agreement. As an example, they quoted that, when the mothers smoked in the house, cases reported this 98% of the time but controls reported this only 85% of the time. Conversely, if the mothers' answers can be believed, only 80% of cases whose fathers did not smoke reported this fact, while 86% of controls whose fathers did not smoke reported accordingly. Questions were also asked regarding the number of cigarettes smoked per day with answers collapsed to none, ≤ 1 pack, 1 pack, and ≥ 1 pack. Regarding mothers' smoking, subjects and mothers agreed exactly 82% of the time, to within one category 95% of the time, and to within two categories 99% of the time. Regarding fathers' smoking, subjects and mothers agreed exactly 49% of the time, to within one category 88% of the time, and to within two categories 97% of the time. Questions were also asked regarding the frequency of smoking in the house with answers collapsed to never, occasionally, and often. Regarding mothers' smoking, subjects and mothers agreed exactly 90% of the time and to within one category 98% of the time. Regarding fathers' smoking, subjects and mothers agreed exactly 71% of the time and to within one category 94% of the time.

Cummings et al. (1989) described the passive smoking histories of 380 never smokers who participated in a study of the respiratory health effects of tobacco smoke exposure conducted at a cancer screening clinic in Buffalo, New York, in 1986. Subjects were asked to report on their exposure to tobacco smoke during childhood years at home, as an adult at home, and as an adult at work. The study also evaluated concordance between subjects' exposure reports and those of surrogates who were questioned regarding the exposure of the subjects. Surrogates for childhood exposure were parents or siblings within 3 y of age of the subject, surrogates for adult exposure at home were mainly spouses, and surrogates for exposure at work were co-workers. For 210 subjects, the surrogate for adult exposure at home was a spouse. The percentage agreement between subject and surrogate was 78.1% for the question, were there any smokers at home, and 89.5% for the question, did the spouse smoke. The intraclass correlation coefficient between responses of subjects and surrogates was 0.70 for the number of smokers at home, 0.93 for the number of years exposed, and 0.85 for the adult home exposure index. The adult home exposure index was computed by multiplying, for each smoker in the household, the number of years exposed by a rating of the severity of the exposure and then summing these scores for all household smokers.

Validation of measurements of recent exposure to ETS

Many studies have been carried out to assess the accuracy of questionnaire measurements of recent exposure to ETS. These studies usually involve the comparison of the questionnaire measurements with various biochemical markers of exposure, particularly cotinine, and/or measurements of exposure obtained from personal air monitors. Measurements of cotinine in saliva, urine, or blood are widely regarded as accurate measures of tobacco use and exposure to ETS during the three to four days prior to measurement.

Wald and Ritchie (1984) reported the urinary cotinine levels in 121 nonsmoking married men according to the reported smoking habits of their wives. The mean urinary cotinine concentration in the men who reported that their wives smoked was 25.2 ng/mL, significantly higher than the mean (8.5 ng/mL) in the men who reported that their wives did not smoke. In addition, they found that the mean duration of reported exposure, both inside the home and outside, was higher for men married to smokers than for men married to nonsmokers. They infer that marriage to a smoker identifies individuals who are more exposed to tobacco smoke in general, not simply from their spouse and conclude that this observation adds plausibility to the results from the epidemiological studies on lung cancer in relation to spouses' smoking habits.

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Jarvis et al. (1984) reported a study in which 100 nonsmoking subjects reported their degree of passive exposure to tobacco smoke over the preceding 3 d and provided samples of blood, expired air, saliva, and urine. The concentration of cotinine in each bodily fluid measured showed significant variation with reported exposure. The mean values for plasma cotinine (ng/mL) were 0.82, 1.81, 2.52, and 1.81 for those who reported no exposure, a little exposure, some exposure, and a lot of exposure, respectively. The corresponding mean values for saliva cotinine (ng/mL) were 0.73, 2.20, 2.80, and 2.63, and for urinary cotinine (ng/mL) were 1.55, 6.50, 8.65, and 9.36.

In Haley et al. (1989), a series of questions regarding ETS exposure was self-administered to nonsmokers and self-reported intensity of exposure was compared with urinary cotinine levels. For both men and women, the mean urinary cotinine level was significantly higher in those who reported exposure in the home than in those who reported no such exposure. The same was true for reported exposure on social occasions but no significant differences were found for reported exposure while traveling or for reported exposure as children.

Coultas et al. (1989) assessed the reliability of questionnaire responses regarding recent exposure to tobacco smoke in the home for a sample of 149 adult nonsmokers recruited in New Mexico in 1986. A structured questionnaire regarding exposure to ETS during the previous 24 h was administered by a trained interviewer to each subject on two occasions separated by approximately four to six months. On each occasion, a urine specimen was collected to allow determination of urinary cotinine levels. The Spearman rank correlation coefficient between the total number of smokers to which the subject was exposed and the urinary cotinine level was 0.24 for the first occasion and 0.21 for the second. The Spearman rank correlation coefficient between the total number of hours that the subject was exposed to cigarette smoke and the urinary cotinine level was 0.32 for the first occasion and 0.29 for the second.

Coghlin et al. (1989) reported a study in which current weekly ETS exposure was measured in nonsmoking volunteers. Three exposure assessment tools were used: a passive nicotine monitor, a baseline questionnaire, and a 7-d diary. An ETS exposure index derived from a baseline questionnaire or from a 7-d diary was developed. All nicotine measurements were closely predicted by a simple linear regression of log nicotine on the index value with $R^2 = 0.98$ for both the questionnaire based index and the diary based index.

Coultas et al. (1990) enrolled 15 nonsmoking volunteers to evaluate the feasibility of measuring personal exposure to ETS at work. During one workshift, subjects wore a personal air monitor to enable collection of air samples for respirable particles (RSP) and nicotine determinations. After the workshift, questionnaires on exposure, together with saliva and urine for cotinine analysis, were obtained. The questionnaire responses were used to provide two measures of exposure to ETS, namely the total number of cigarette smokers to which the subject reported being exposed during the workshift and the total number of hours for which the subject reported being exposed to ETS during the workshift. Spearman's correlation coefficient was 0.44 between total number of smokers and RSP, and 0.53 between total number of hours exposed and RSP. The corresponding correlation coefficients were 0.62 and 0.54 for nicotine, 0.39 and 0.57 for urinary cotinine, and 0.63 and 0.45 for salivary cotinine.

Cummings et al. (1990) obtained reports of recent exposure to ETS and urinary cotinine levels for 663 never-smokers and ex-smokers who attended a cancer screening clinic in Buffalo, New York, in 1986. The correlation between urinary cotinine level and the number of reported exposures to ETS was 0.23. Having controlled for the number of exposures, only the ventilation characteristics of the exposure location was found to be significantly related to cotinine levels. The mean cotinine value was 6.22 for the 162 subjects who reported no exposure, 7.75 for the 208 subjects who reported 1 or 2 exposures, 9.75 for the 152 subjects who reported 3 to 5 exposures, and 12.50 for the 141 subjects who reported 6 or more exposures. The authors reported considerable overlap in the cotinine readings of the 4 exposure groupings.

In Proctor et al. (1991), 52 nonsmoking British women were recruited to wear personal monitors for nicotine over a 24-h period in the autumn of 1989. The subjects also supplied samples of saliva for cotinine analysis, and answered questions regarding lifestyle and exposure to ETS. The correlation between the reported number of cigarettes smoked close to the subject was 0.50 with nicotine and 0.63 with salivary cotinine. For subjects living in smoking households, the correlation between the reported number of cigarettes smoked close to the subject at home was 0.80 with nicotine and 0.69 with salivary cotinine. All subjects were also asked to assess their exposure to ETS scaled from 1 (none) to 5 (extreme) during the sampling period. The authors reported that these assessments did not correlate well with either nicotine or salivary cotinine levels.

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In Becher et al. (1992), nonsmoking females, age 35-65 y from Bremen, Germany (91 women), and Opole, Poland (98 women), were interviewed about their recent passive smoking exposure. Urine samples were obtained at the time of the interview to determine the concentration of cotinine as an indicator of tobacco smoke exposure. For German women, the mean cotinine level was 17.96 for those who reported exposure to ETS from their husband and 5.50 for those who reported no such exposure. The corresponding values for Polish women were 16.28 and 6.08, respectively. For German women, the mean cotinine level was 8.82 for those who reported exposure to ETS in the workplace and 8.55 for those who reported no such exposure. The corresponding values for Polish women were 13.38 and 8.10, respectively. The Spearman correlation coefficient between the cotinine level and the time-weighted sum of exposures was 0.66 for German women and 0.62 for Polish women. A regression model, using age and the time-weighted indices of exposure from the husband, from other people at home, in the workplace, in vehicles, and in other places, produced an R^2 value of 0.62 for German women and an R^2 value of 0.40 for Polish women. When the variable "husband is a smoker" was added into the model, however, the fit improved for the Polish group (R^2 = 0.48), whereas for the German group, the improvement was only marginal. The authors concluded that this difference indicates that passive smoking exposure due to the husband is less correctly reported in the Polish group. Two divisions of subjects into four groupings were considered. The first was based on the quartiles of the distribution of time-weighted exposure from all sources and the second was based on the quartiles of the distribution of urinary cotinine. Taking the cotinine grouping as indicative of true exposure, the following distributions for subjective assessment of exposure were found:

		Reported category			
		1	2	3	4
	1	54	37	7	2
True	2	28	26	38	8
Category	3	17	33	29	21
	4	0	6	27	67

So, for instance, 2% of those whose cotinine value indicated that their exposure was relatively low were classified as being in the highest quartile of reported exposure.

Delfino et al. (1993) obtained salivary cotinine readings from 258 nonsmoking bank employees who simultaneously answered questions detailing their exposure to second-hand smoke within the previous three days. Exposure models were created to take into account the number of smokers nearby, length of time in their presence, half-life of cotinine in bodily fluids, level of aversion to cigarette smoke, and time of year. All models, including consideration of intensity and duration of exposure combined, succeeded in explaining only 16% of the variation in log cotinine levels.

Forastiere et al. (1993) evaluated the strength of association between urinary cotinine and questionnaire data on passive smoking among 542 adolescents. The group of 103 individuals with cotinine levels higher than 30 ng/mL were compared with the 439 individuals with cotinine levels lower than 30 ng/mL using logistic regression. After a forward stepwise regression procedure, the final logistic model included the following variables: log of urinary creatinine, subject's perception of passive smoking at home, maternal smoking (parental report), house crowding, and the interaction between the last two variables. Subjects whose probability of being exposed was higher than 0.60 were classified as being in the group with cotinine higher than 30 ng/mL. This method of classification yielded a sensitivity of 86.3% and a specificity of 70.5%.

In Marbury et al. (1993), the relationships among three methods of measuring exposure to ETS, questionnaires, urinary cotinine, and a passive monitor for ambient nicotine, were investigated in a study of 48 children in Minnesota in 1989. Activity room concentration was highly correlated with the total number of cigarettes smoked in the house (r = 0.86). Regression analyses of activity room nicotine and urinary cotinine showed that the indicator variables for which of the parents smoked (neither, mother only, father only, both) were better predictors than the total number of cigarettes smoked in the house.

O'Connor et al. (1993) compared three methods of measuring exposure to ETS in a sample of 415 self-reported nonsmoking pregnant women - questionnaire, personal monitor, and urinary cotinine. With the monitor as standard, the questionnaire-based classification of a subject as exposed or unexposed had a sensitivity of 70.8% and a specificity of 59.9%. The Spearman correlation coefficients for air nicotine concentration with questionnaire variables were: 0.41 for total duration of exposure, 0.34 for duration of exposure at home, 0.18

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for duration at work, 0.28 for social duration, 0.35 for number of smokers at home, 0.34 for number of cigarettes smoked at home, 0.16 for number of smokers at work, and 0.17 for number of cigarettes smoked at work.

Bono et al. (1994) reported a study to evaluate the relationship between ETS exposure as measured by questionnaire and urinary cotinine in 120 nonsmoking schoolchildren. The mean cotinine level was 21.42 ng/mL for the 39 children reporting no exposure to ETS at home, 23.81 for the 81 children reporting some exposure to ETS at home, 24.75 for the 14 children reporting exposure to ETS at home from the mother only, and 28.37 for the 28 children reporting exposure to ETS at home from the mother and others.

Kemmeren et al. (1994) used a questionnaire to elicit the number of hours of passive exposure to ETS for a large number of volunteers. The unexposed group consisted of 55 nonsmokers from the lower tail of the distribution of self-reported passive exposure. The exposed group consisted of 50 nonsmokers from the upper tail of the distribution of self-reported passive exposure. There was considerable overlap in the cotinine values of the two groups. The mean plasma cotinine concentration was 0.6 ng/mL for the unexposed group and 1.6 ng/mL for the exposed group. The correlation between hours of passive smoke exposure and plasma cotinine was 0.42. However, the correlation between hours of passive smoke exposure and plasma cotinine was 0.14 within the exposed group and 0.24 within the unexposed group. The cut-off value which misclassified the fewest subjects in relation to self-reported exposure was 1.1 ng/mL of cotinine. The percentage of the unexposed group correctly classified was 89%. The percentage of the exposed group correctly classified was 56%.

Ronchetti et al. (1994) reported the relationship between salivary cotinine levels and household smoking habits for a group of 109 nonsmoking Italian schoolchildren, age 9-14 y. For the 56 subjects who reported no cigarettes smoked in the household, the percentage with detectable cotinine levels was 14% and the geometric mean of cotinine concentration was 0.66 ng/mL. The corresponding values were 10% and 0.66 ng/mL for the 10 subjects who reported 1-19 cigarettes smoked in the household, 24% and 0.91 ng/mL for the 17 subjects who reported 20-39 cigarettes smoked in the household, and 67% and 1.77 ng/mL for the 26 subjects who reported more than 40 cigarettes smoked in the household.

In Phillips et al. (1994), the ETS exposure of 255 nonsmoking subjects was assessed by several methods. Each subject wore a personal air sampler for 24 h, answered a questionnaire about ETS exposure, and provided saliva samples for cotinine analysis before and after the monitoring period. Saliva cotinine levels showed poor correlation ($R^2 = 0.06$ for pre, $R^2 = 0.14$ for post) with 24 h ETS particle exposure and also showed poor correlation ($R^2 = 0.07$ for pre, $R^2 = 0.13$ for post) with 24 h nicotine exposure. Subjects were divided into three groups based on whether their spouse did smoke, did not smoke, or they had no spouse. Both the subjective assessments of the intensity of ETS exposure and measured exposure levels indicate that the ranking of exposure is smoking partner > no partner > nonsmoking partner. However, on both scales, there is considerable overlap among groups. For instance, 29% of subjects with a smoking spouse were exposed to less than the mean ETS particle level of subjects with a nonsmoking partner. Subjects were divided into five groups based on their subjective assessment of the intensity of their exposure to ETS (none, low, moderate, high, and very high). Based on directly measured exposure levels, the authors concluded that subjects were able to assess an exposure of none or low well but that there was considerable variation in the direct measurements corresponding to the higher grades of subjective assessment. For instance, some subjects who reported their exposure as high had less directly measured exposure than other subjects who reported their exposure as low.

THE TUNSTALL-PEDOE STUDY

Tunstall-Pedoe et al. (1995) carried out a cross sectional random population survey involving 2278 nonsmoking subjects to explore the relationship between ETS exposure and coronary heart disease (CHD). Each subject was sent a questionnaire to complete and a clinic appointment. The questionnaire included the standard Rose angina and possible infarction questionnaire, the Medical Research Council cough and phlegm questionnaire, and questions on prior medical diagnoses. In addition, subjects were asked to answer the question, "Have you been exposed to tobacco smoke from someone else in the last three days?", with possible answers: "4 - yes, a lot; 3 - yes, some; 2 - yes, a little; 1 - none at all".

In analyses comparing the group who answered "yes, a lot" to the ETS exposure question with the group who answered "none at all", significant positive associations were found for CHD (OR = 1.6, 95% CI [1.1, 2.4]), chronic phlegm (OR = 2.3, 95% CI [1.4, 3.9]), and chronic cough (OR = 2.3, 95% CI [1.3, 3.9]).

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In the course of the clinic visit, subjects were asked to provide a blood sample from which serum cotinine readings were obtained. Though cotinine has some advantages as a marker, Phillips et al. (1994) argued that it is not a completely valid marker of true exposure. People vary in the extent to which they can metabolize nicotine to cotinine and in any case nicotine may not be the best indicator of exposure to "relevant" smoke constituents. In spite of these reservations, cotinine is the most widely used marker of ETS exposure (Riboli et al. 1990).

Replies to the question on passive smoking produced four unequal groups with 618 subjects giving reply 1, 814 giving reply 2, 554 giving reply 3, and 292 giving reply 4. Cut points were chosen in the serum cotinine results to give groups which corresponded in size to their questionnaire equivalents. Taking the cotinine grouping as indicative of true exposure, the following distributions for subjective assessment of exposure are found:

		Reported category			
		1	2	3	4
True Category	1	38	36	18	8
	2	29	37	23	11
	3	19	36	30	15
	4	12	31	32	25

So, for instance, 8% of those whose cotinine value indicated that their exposure was relatively low replied "yes - a lot" when asked to provide a subjective assessment of their own exposure. Clearly the correlation between the two measures of exposure was poor.

In analyses comparing the highest cotinine group with the lowest cotinine group, the significant positive associations noted above disappeared: CHD (OR = 1.2, 95% CI [0.9, 1.7]), chronic phlegm (OR = 1.2, 95% CI [0.7, 2.0]), and chronic cough (OR = 1.1, 95% CI [0.6, 1.9]). The authors conjectured that biased reporting of ETS exposure may account for this discrepancy and concluded that "the validity of different measures of tobacco smoke exposure needs further investigation". This study should serve to alert epidemiologists to the need for care when interpreting associations between disease and subjective exposure assessments.

DISCUSSION

The Fontham study has been widely quoted as the most comprehensive and best designed study to establish an association between lung cancer and exposure to ETS. The conclusions of the study are presented as if subjects were able to accurately report their level of ETS exposure over long periods of time. However, the reports of the study suggest that no formal attempt was made to validate the questionnaire used in the study.

The literature reviews presented in this paper make it clear that exposure to ETS is not accurately measured by subjective evaluations communicated via questionnaire. The inaccuracy is evident in situations where recent exposure is being measured and may reasonably be expected to be even greater when long-term exposure is at issue. One might expect there to be no inaccuracy in a wife's response to a simple question such as: Did your husband smoke? However, reported disagreements between husbands and wives and between wives and children imply that some inaccuracies do occur.

Inaccuracies do not necessarily invalidate the conclusions that may be drawn from a case/control study. In a situation where errors occur randomly and in a similar fashion for both cases and controls, the only effect of such errors is to reduce the power of the study and to make it more difficult to establish an association between exposure and disease. However, in a situation where the pattern of errors is different for cases than it is for controls, there is considerable potential for the errors to produce misleading conclusions. This is the phenomenon known as differential recall bias.

The study by Tunstall-Pedoe et al. (1995) strongly suggested that differential recall bias occurs in practice. In that study, associations found to exist between subjective exposure assessments and disease were not found when exposure assessments via cotinine were used instead of subjective assessments. The authors suggested that differential recall bias may be the sole reason why associations were found when subjective exposure assessments were used.

Remarkably little effort has been made to address the magnitude of differential recall bias. The only study found which compared cases and controls in terms of the accuracy of their responses to questionnaires was that of Sandler and Shore (1988), where evidence of modest recall bias was presented. It seems reasonable to expect that the increased media publicity given to claims of an association between ETS exposure and lung cancer would induce over-reporting of ETS exposure among lung cancer cases relative to respondents who were not suffering from lung cancer. There is a need for a much greater research effort to determine if differential recall bias occurs and, if so, to quantify the magnitude of this bias.

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The methods for assessing sensitivity of findings to differential recall bias described in Barry (1996) are applicable to case/control studies of exposure and disease. The findings of such studies would be greatly strengthened if the extent of the differential recall bias required to insubstantiate them was considerably greater than that found in the studies of differential recall bias recommended in the previous paragraph.

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